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Typhoid Fever.*

BY

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ON SOME FORMS OF PARALYSIS AFTER TYPHOID FEVER.¹

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In a short communication relating chiefly to some of the surgical sequels of typhoid fever, Sir James Paget says:

"I do not feel competent to deal with the question whether each fever has, as seems probable, its own proper sequels, and in this sense, though perhaps in less degree, as specific as in its fever-period, but I have not yet seen either periostitis or local paralysis of muscles after any other than typhoid fever, or a fever which I suppose to be closely related to it, and I have not seen after typhoid any corresponding number of cases of large lymph glands, diseased joint, or other diseases of mere debility, such as may follow any acute illness."

And he quotes the remark of Dr. Baly, to the effect that "a book on the diseases of convalescence might be one of the most valuable that any physician could write." A consideration of the truth of this remark has led me to lay before the Association a few notes upon the important subject of paralysis following typhoid fever.

It is an interesting question how far these so-called sequels or well-defined disorders occurring during the convalescence from fevers and other acute diseases are special or confined to certain of them only, and how far others may occur in a similar relation to several of them. Certainly, in many instances, the latter is found to be the case, as, *e. g.*, otitis media after both scarlatina and measles; whilst, again, some peculiar local derangements seem to follow in the wake of a particular fever, and to bear no similar relations to any other, *e. g.*, paralysis of the palate following diphtheria. So with periostitis after typhoid, which may occur in many situations. I have seen it several times on the tibia, once leading to severe necrosis, often on the ribs, and twice on the surface of the sternum. So with the affections of the nervous system, the most common form being of the muscles supplied by the peroneal or anterior tibial nerve, whilst more extensive paralyses are decidedly uncommon. The very peculiarities of the parts affected, and the frequency with which they are attacked in preference to all others, show certainly a predilection for them which must give the stamp of specificity.

¹ Read before the Association of American Physicians, Washington, 1888.

It is not to be wondered at that the nervous system suffers in many ways after the prolonged disturbance to which it is subjected during a siege of typhoid fever. Indeed, it would be remarkable if derangements of the nervous functions were not often witnessed under these circumstances. In a certain proportion of all cases of this fever, the incidence of the poison seems to be upon the nervous system, as is evidenced by the occurrence of cephalalgia, insomnia, delirium, tremors, and debility. As a sequel of these profound and prolonged disturbances in the nervous system all patients who have thus suffered are left in a condition of very marked asthenia, and it is a long time before they are in a condition to bear any, even moderate, amount of either bodily or mental fatigue. These signs of exhaustion of the nervous system are constant, and generally in proportion to the severity of the fever, and the degree to which its violence has fallen upon that portion of the mechanism. But, apart from such general functional diminution, there are, in exceptional cases, signs that certain isolated, and often strictly localized, portions of the nervous system have suffered out of all proportion to the rest, and in them the impairment of function may reach a high degree. Thus, the cerebral cortex may be the part chiefly affected, and we observe, even after full convalescence has set in, that the mental functions remain imperfect and hallucinations and delusions may persist for a very considerable time—or, as concerns us most at present, some part of the spinal cord, or some one or more of the spinal nerves, exhibits altered function.

It is, however, a fact that, although these sequential paralyses are more frequently observed after somewhat severe attacks of typhoid, yet they do not specially occur when the fever has been marked by an excess of nervous disturbance. Nor can any such predisposing cause as the influence of country or of a neurotic temperament be shown to play any part. The nervous phenomena, almost invariably, are both motor and sensory, presenting paralysis with either pain or anæsthesia. This has always been observed in *spinal* nerves, and has not been seen in any of the mixed (motor and sensory) central nerves. According to Nothnagel (*Deutsch. Archiv für klin. Med.*, 1872), from an examination of recorded cases, the following is the order of frequency of these affections: 1. The parts supplied by one nerve or branch of a nerve, with special predilection for the ulnar and the peroneal. 2. Paraplegia, generally confined to the lower extremities, but not very infrequently involving an upper extremity—sometimes both arms and legs—sometimes one side more than the other. 3. Less frequently, one extremity, either upper or lower, or two extremities in crossed order. 4. Simple alterations of sensibility; if looked for, these will be found *very frequently*, though not so striking as the cases with combined paralysis and anæsthesia. They are observed chiefly in the lower extremities and especially in the feet.

From a consideration of many cases of paralysis after this fever, it

may be inferred that the mode in which it is produced is by no means always the same; that, indeed, the effect of the poison is exerted sometimes upon some of the great nervous centres, especially of the spinal cord, and at other times upon the structures of some special and individual nerve or nerves. Jaccoud, *e. g.*, speaks of paraplegia being due to *œdematous infiltration* of the parts in the vertebral canal, and is of opinion that even prolonged *exhaustion* of these centres without organic lesion may be the cause of a motor loss enduring even for a considerable time. Prof. Biermer relates an important case, in which he finds difficulty in deciding between acute poliomyelitis anterior and multiple degenerative neuritis, there having been pain, motor paralysis, good sensation, diminished electrical reaction, atrophy, and bedsores, a tedious illness of several months, and, finally, complete recovery. As the tendency in all these cases is toward recovery their pathology is necessarily scanty and founded upon what is better known about recognized lesions whose symptomatology is somewhat analogous.

The commonest variety of post-typhoid paralysis is that which is confined to the district supplied by one nerve, *e. g.*, the ulnar, peroneal, or the circumflex. Here, it is extremely probable that two distinct pathological conditions may exist. There may be a true interstitial neuritis or there may be an acute parenchymatous change in the nerve elements. The two varieties are separated from each other mainly by the presence or absence of *pain*—this symptom being a marked feature in the former, owing to the pressure exerted upon nerve-filaments, and being absent in the latter where this condition does not exist; the main feature, however, the loss of power in the supplied muscles, being equally present in them both. It has been suggested by a recent writer (Thomson) that a similar acute parenchymatous degeneration is to be looked upon as explaining that common post-febrile paralysis, the diphtheritic, and the view has much to commend it.

It is not unusual after typhoid fever of considerable severity to find a definitely enfeebled condition of the lower extremities persisting for some time, and sometimes a person never entirely recovers his capacity for walking long distances. Such parietic cases have never been specially studied, but it is probable they would, if any should, fall under the head of defective innervation from prolonged exhaustion of the nervous centres.

The most important, however, of cases of post-typhoid paralysis are those of paraplegia, confined sometimes to the lower extremities and sometimes involving these together with some other parts. Well-marked cases of this kind are sufficiently unusual, and one may see several hundreds of cases of typhoid fever without meeting with a single example. Not much has been written upon it, and in some articles very dissimilar conditions are probably included under the same description.

The following case is a striking instance of a very severe form of paraplegia directly resulting from typhoid fever and still ending in complete recovery.

Julia L. was brought by the ambulance to the Montreal General Hospital on the 8th November, 1887. The following account of her illness was obtained from her attending physician. She complained on the 1st September of headache, feverishness, pains in the limbs, and a short hacking cough. After one week took to her bed. Was first seen by her physician on 12th September. At this time she had a severe cough with whitish expectoration. Moderate distention of abdomen, and gurgling in right iliac fossa. Temperature 102° F. The disorder ran the ordinary course of a moderately severe attack of typhoid fever without complication. Patient remained in bed until October 25th, when she got up, went about the house for three or four days and was apparently convalescent; appetite good, sleeping well, and gaining strength rapidly. On October 29th temperature rose to 102° F. and she began to suffer from severe pains in the legs. The lower extremities were very sensitive, the patient not being able to bear any motion of the bedclothes. The legs were flexed on the thighs and the thighs on the pelvis, any attempt at extension being very painful. At this time had incontinence of urine for one day. The skin over the left trochanter and sacrum very soon became red, and in two or three days a large slough, about two inches square, had formed in both these situations. The skin over the right trochanter also became reddened but did not slough. The temperature soon became normal and remained so.

On admission, much emaciated and anæmic; face pale, eyes brilliant, pupils dilated and equal; expression anxious and haggard; skin dry and harsh, cool; tongue clean and moist; abdomen retracted, not tender on pressure. Lies on right side with legs and thighs flexed. Some œdema of left foot. Numerous small purpuric spots on both legs from knees downward. Great wasting of muscles of both lower limbs, thigh and leg; both knees are rigidly bent; any movement to relieve the contraction is extremely painful. Marked paresis in all attempted movements of foot, leg, or thigh. She is unable to hold up either leg from the bed without support. Much tenderness on pressure upon the muscles of the legs or thighs. No anæsthesia. The skin over the two malleoli somewhat reddened. Plantar and abdominal reflexes present. There is a large bed sore over the lower part of the sacrum, about two inches square, covered with a hard, black slough and with inflamed edges. There is a similar slough on the left trochanter, and the skin over the right trochanter is reddened but unbroken. Pulse feeble and rapid, but regular. Temperature normal. In lungs, many moist sounds heard at both bases, chiefly in the left. Urine contained a small amount of albumin. Complaints of pain over the bedsores and weakness. Eats well. Sleeps soundly. Urine and feces passed naturally. Is very feeble and resents being moved or stirred.

She was put upon a water bed and the bedsores poulticed, and was given quinine, good diet, and a small allowance of stimulants. The slough soon separated and the sores were dressed with balsam of Peru and iodoform; they slowly granulated and finally healed. The contraction of the knees obstinately remained. In February, the legs were gradually extended by means of weights and pulleys until they became

quite straight. During this time, motor power was slowly returning and pain quite disappeared. After a time, she could stand upright, and perform the flexion and extension movements of the feet and the legs. Improvement was then rapid, and she was discharged March 1, 1888, able to walk nicely about the ward. She has since remained well and strong.

During apparent convalescence, there occurred severe pain in the limbs, marked tenderness of skin and muscles, no anæsthesia, diminution of superficial reflexes, loss of electrical contractility, extensive bedsores, loss of power to a great extent but never complete, contractures, no disturbance of sphincters. The case looked very unpromising and it was only the known tendency of these cases toward recovery and the probability that the lesion was really in the nerve-trunks, and not in the spinal cord itself that enabled a somewhat favorable opinion to be entertained.

The second illustrative case is still more remarkable inasmuch as it presents an example of unusually extensive paralysis after typhoid—the nervous disorder involving all the limbs and, in addition, the palatal muscles after the manner of diphtheritic palsy.

Ernest H., æt. fifteen years, was admitted under my colleague, Dr. Wilkins, into the Montreal General Hospital on the 26th of March, 1888, suffering from typhoid fever of a few days' standing. The case presented no unusual features, and was of a rather mild type. On the twenty-fourth day of illness the temperature was 99° , and in a few days later it reached and continued at the normal figure. About this time he found that he spoke with some difficulty—that is to say, the effort required for speech was greater than it should be; it was noticed that his speech had a nasal intonation. There soon followed a sense of numbness in the ends of the fingers and in all the limbs; the weakness being greater upon the left side than the right. By the 4th of May he lay in bed without power to raise either leg from the bed, the head and arms were raised with great difficulty, and the grasp was very feeble. There were no pains in the extremities. The voice was quite nasal, speech was difficult, and swallowing required an effort and occasionally produced regurgitation. The velum palati was relaxed and insensitive. Knee-jerk absent in both legs. Tactile sense good. Muscles of lower extremities react normally to the faradic current, but somewhat slowly to the galvanic current. Muscles of the upper extremities react three times as strongly on the left side as on the right side. Eyes examined by Dr. J. J. Gardner: "Pupils react to accommodation and light, but somewhat lazily to light. Accommodation unimpaired. External ocular muscles act well, no weakness of internal recti. Fundus normal." He remained in this condition of extensive paresis, under treatment by means of galvanism and strychnia for several weeks, being removed by his friends on the 27th of June, 1888. At this time the limbs had improved in power to a moderate extent, and the nutrition was better; the voice was less nasal.

Gowers (*Diseases of the Nervous System*, vol. ii.) alludes to similar cases, but mentions that, as diphtheria is known sometimes to accompany typhoid fever, so a paralysis in reality of diphtheritic nature may be observed as a sequence of such a compound attack; and the question may be raised whether H. did not have diphtheria. There is no proof that he did have throat affection of any kind; and, on the other side, he was, during the whole time, under skilled attendants in a hospital, and nothing was ever noticed or complained of which led to an examination of the fauces. It may, therefore, I think, be safely held that no diphtheria complicated the case. I would also recall, as strongly corroborative of the same view, that there was no disturbance of accommodation, and no albumin in the urine.

Although actual observations are not very numerous showing presence of the lesions of peripheral neuritis, and the absence of change in the spinal centres in cases in which post-typhoid paralysis has existed, yet they are sufficient to substantiate the occurrence of such a disorder. Indeed, in the light of the somewhat remarkable observations recorded by Pitres and Vaillard (*Revue de Médecine*, t. v., 1885), we may well be surprised that the clinical evidences of neuritis are not more frequently met with. These writers made careful and extensive histological examinations of peripheral nerves in various parts of the body in cases of typhoid fever which had proved fatal from various causes, but in none of whom there had, at any time, been evidences of nervous lesion or disorder. The result was, in every case, to find microscopical evidence of well-marked changes in the structure of most of the nervous trunks and their branches, whilst the actual nerve-roots and the spinal centres remained unaltered. And the question is raised, "How frequently in reality does such neuritis occur?" In the examples recorded there had been nothing from a clinical standpoint to suggest its existence, and they were selected quite by chance. Does it occur both in fatal cases and in those mild or more severe cases which end in recovery? We are led to think the latter possibly true, owing to the frequency with which more or less marked evidences of sensory, motor, or trophic disturbances are met with as sequences of typhoid fever.

Is this neuritis set up by the elevation of temperature? or by the general derangement of nutrition? Or may the typhoid poison act directly upon the nervous fibres, irritate these, and ruin their structural integrity—determine, in fact, a parenchymatous neuritis? This neuritis, it is stated, attacks not only the superficial branches which innervate the skin, but also the larger and deeper trunks. May it not, therefore, be that the disturbances in them are to blame for many of the symptoms generally observed in typhoid fever: cutaneous hyperæsthesia, muscular hyperæsthesia, wandering or localized pains in the limbs, etc., symptoms which are often attributed, perhaps rather gratuitously, to irritation of the

meninges, or of the spinal cord? The truth would seem to be that when a neuritis of this kind is mild in character and affects a few only of the fibres of any particular nerve, no appreciable symptoms are produced thereby, or, at any rate, such slight phenomena as it may induce are lost in the complex symptomatology of the pyrexia; and that, for some obscure reason, these nervous lesions sometimes become exaggerated, extend to still other bunches of fibres, and then give rise to the well-marked symptoms due to a diffuse or a localized neuritis: sensory, motor, and trophic disorders, confined to the region supplied by one or several nerves, according to the distribution of the determining lesions.

As regards the *etiology* of this affection, some authors have regarded it as due to the anemia which must necessarily result from a long febrile illness. That such is not the case is proved by the actual condition of many of the patients who have shown no such degree of blood-impoverty as would be at all sufficient to account for serious disorder of the nervous centres or their branches. The series of cases produced by Nothnagel were selected from the barracks, and were all young, vigorous, and full-blooded men. The effects of any such anemia would surely be more general, involving loss of energy in movements, but not a true paralysis. It could not possibly produce the clearly defined paralysis affecting special nerves as we find after typhoid fever. The other hypothesis which has been suggested is that the disorder consists essentially in a degeneration of the affected muscles. That this cannot be, is sufficiently obvious from the extensive sections of the body which may be simultaneously involved as—*e.g.*, both lower extremities, or these together with an upper extremity; from the constant presence of pain at some period, and often severe; and from the existence of anesthesia.

Post-typhoid paraplegia generally sets in gradually and disappears gradually, but, in rarer examples, it comes on suddenly and passes off either suddenly or, at any rate, very quickly. Such a case is that reported by Ollivier, which led him to infer the existence of a true, but temporary, congestion of the spinal cord. There is nothing impossible about the hypothesis, and it seems necessary in order to explain such a case, but certainly does not apply to the more common forms of gradual development. Nothnagel suggests that actual spinal hemorrhage is more probably here present, and that meningeal hemorrhage has, in reality, been often found in fatal cases of typhoid fever.

The predilection for certain special groups of muscles observed in the paralyzes of plumbism and of diphtheria does not exist in the same degree after typhoid fever. In it there is no rule, and, to explain it, we must look for some cause capable of making its influence felt in an infinite variety of situations: the ulnar or the peroneal nerve, a plexus of nerves, both lower extremities, an upper, together with the opposite lower, extremity. At the same time, all degrees of intensity may be

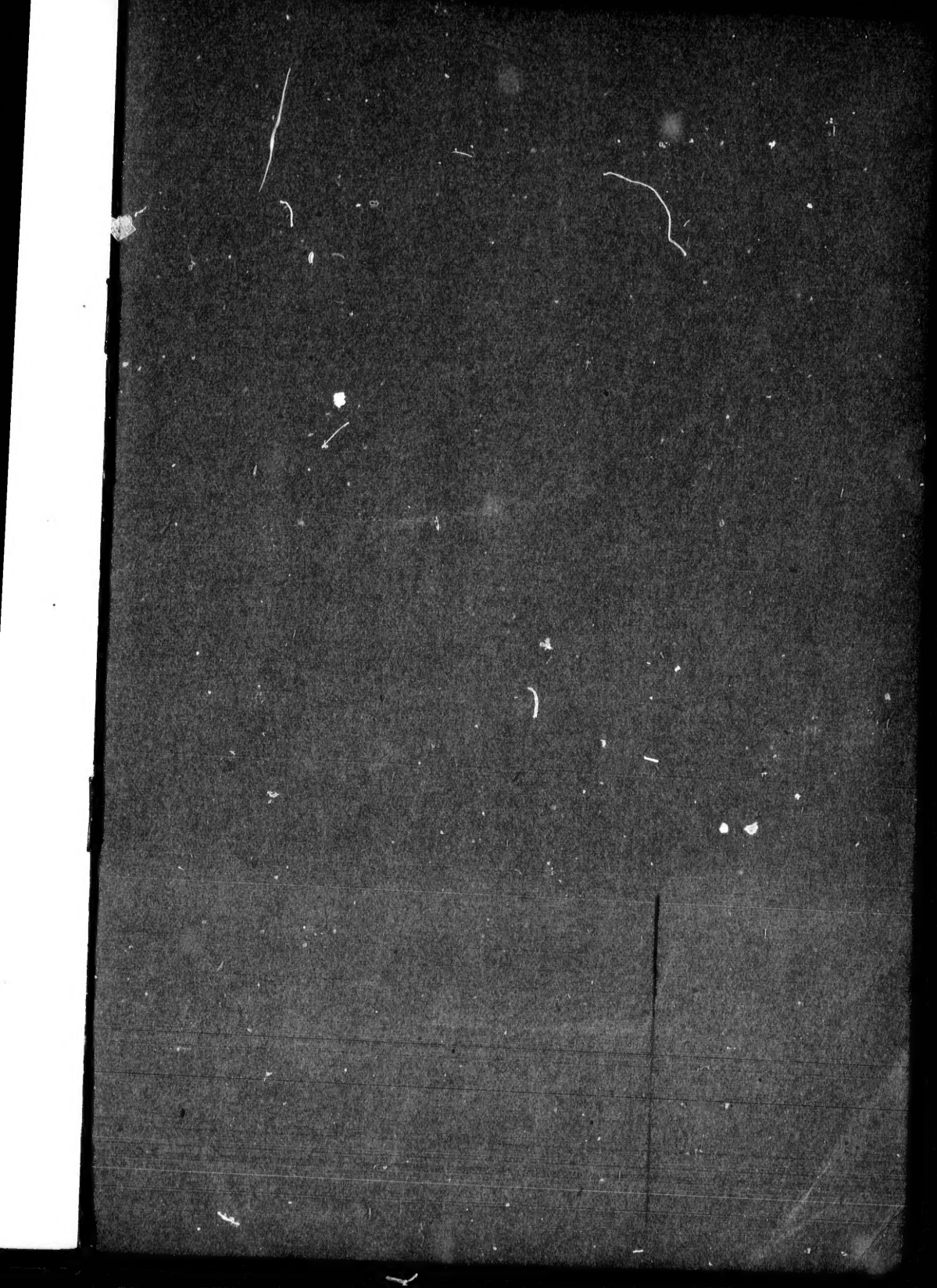
observed, from the slightest paresis up to complete abolition of function.

The paralysis generally develops slowly and gradually, and it commonly advances to a considerable degree before being arrested. Arrived at this stage, it may be expected to recede spontaneously or under the influence of treatment, and the case to end in complete restoration of function. A more unusual course is that in which, as before, a slight, or very moderate degree of weakness is gradually developed, and then suddenly a great change occurs, and a perhaps complete paralysis sets in. Even here, the usual result is gradual return of power. In either case, there are exceptional instances in which the paralysis will be permanent, even in spite of early and efficient treatment.

The first symptom is generally pain, and this may precede any motor defect by some considerable time. Pretty often, the pain and the commencement of the paralysis are noticed about the same time.

The cutaneous sensibility is nearly always diminished, and these sensory disturbances—*i. e.*, pain and anæsthesia—are amongst the most constant phenomena of this affection. In severe and well-marked cases trophic changes are seen, consisting chiefly in a greater or less degree of atrophy. There is diminution of electrical contractility, both to the galvanic and to the interrupted current.

From a consideration of these general features Nothnagel strongly argues that the condition is essentially the same as one of traumatic paralysis when the injury has been done by some gradually compressing cause. We must, in that case, look for some pathological cause which is capable of acting in this way. Certain it is that, in fatal cases of diphtheritic paralysis (which has many affinities with that under consideration) there has been found evidence of thickening of the anterior and posterior roots of the involved nerves, especially in the neighborhood of their point of junction; and also of diphtheritic infiltration within the nerve-sheaths. It is probable that there would be found, in an early stage, proliferation of the connective tissue, and that the subsequent contraction which always sets in under these circumstances would keep up the compression of the nerve-filaments. The same hypothesis would apply to examples of extensive—or even quite limited—areas of anæsthesia.



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